# THE SEPTO-HIPPOCAMPAL PATHWAYS AND THEIR RELEVANCE TO HUMAN MEMORY: A CASE REPORT

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## INTRODUCTION

The medial septal/diagonal band complex appears to be a crucial structure for the normal functioning of the hippocampal formation ( $HF^1$ ) (Olton, 1990). The septal nuclei which, contrary to the prevailing opinion, are well developed in human brain (Andy and Stephan, 1968) are poorly individualized cell groups whereas the vertical limb of the nucleus of the diagonal band, the other component of this basal forebrain complex, is an aggregation of rather large cells.

Electrical stimulation of the medial septum in rats produced significant increases in bilateral hippocampal regional cerebral blood flow (rCBF) whereas other brain regions were not influenced. The responses of increased rCBF were confirmed for the regions that receive cholinergic nerve projections from the medial septum indicating that the responses may be related to activation of the cholinergic fibres (Adachi et al., 1990).

The septal region is a principal site for Acetylcholine-Dopamine (ACh-DA) interaction. In septal nuclei DA ligands have been found to affect the activity of hippocampal cholinergic projections arising from the medial septal/diagonal band complex (Robinson et al., 1979; Swanson, 1982). Particularly this ACh-DA interaction may be necessary for the maintenance of accurate cognitive performance (Levin et al., 1990).

Damage to the medial septal area in rodents produced severe deficits in various tasks testing spatial working memory (Beatty and Carbone, 1980; Kelsey and Landry, 1988; Brito et al., 1983; McLamb et al., 1988).

In humans, lesions of the septal region have repeatedly been described in cases of ruptured aneurysms of the anterior communicating artery (ACoA) (Gade, 1982; Alexander and Freedman, 1984; Damasio et al., 1985; Corkin et al., 1985).

A PET study of two patients with amnesia after rupture and repair of ACoA aneurysm indicated a secondary remote effect on mediobasal temporal structures (Volpe et al., 1984). Thus, an indirect effect on the HF may finally be responsible for memory dysfunctions observed in these basal forebrain cases.

<sup>&</sup>lt;sup>1</sup> Following the definition proposed by Amaral and Witter (1989) we subsume under the hippocampal formation four relatively simple cortical regions: (1) the dentate gyrus, (2) the hippocampus proper (CA3, CA2, CA1), (3) the subicular complex and (4) the entorhinal cortex.

Lesions within the human septal region are not restricted to nuclear structures but may include a multitude of neuromediator-containing fibres traversing this region, among them NE- or 5-HT-containing fibres from the brainstem on their way down to the HF (Nieuwenhuys, 1985).

A case underlining the relevance of an intact septal region to explicit memory and learning functioning in humans was published by Phillips et al. (1987). Following the repair of a ruptured ACoA aneurysm their patient developed anterograde amnesia, apathy (loss of volition) and partial diabetes insipidus. Postmortem examination of the brain revealed infarctions in the supply area of the ACoA with destruction of the septal gray, the nucleus of the diagonal band, the nucleus accumbens, a small rostro-ventral portion of the globus pallidus, and a central segment of the anterior limb of the internal capsule. The most caudal extent of the lesion spared the anterior commissure and the nucleus basalis of Meynert. The remainder of the brain was unremarkable.

A second case hinting at the septal region as a potential memory-relevant neural structure was recently published by Berti et al. (1990). Their patient developed anterograde amnesia after transcallosal excision of a tumour of the septum pellucidum. Neuroimaging procedures did not show any major lesion of brain areas traditionally associated with amnesia. Over a period of four months after surgery the patient had not shown any improvement in standard memory tests apart from paired-associates learning. In our opinion the lesion included the upper part of the septal region damaging the septal nuclei but presumably sparing the nucleus of the diagonal band.

Fibre projections interconnecting the medial septal/diagonal band complex and other traversing pathways with the HF follow two different routes. The majority of fibres pass through the fornix-fimbria system, smaller contingents enter the supracallosal longitudinal striae (as part of the supracommissural hippocampus), and the cingulate bundle. These parallel fibre bundles constitute the *dorsal route*. In some species (e.g. in cats) septal fibres travel to cranial parts of the HF along a *ventral route* through the ansa peduncularis and the amygdala (Gage et al., 1984).

If it is true that the interaction between the septal region and HF is indispensable for the maintenance of normal memory and learning mechanisms in humans, a disruption of some combination of these septo-hippocampal fibre projections, especially a disruption of the dorsal route, should crucially deteriorate explicit memory functions. The aim of this paper is to present a case who may credit to this assumption.

## CASE REPORT

#### Patient

On September 16, 1990 D.C., a 25-year-old, right-handed car mechanic awoke with a splitting headache. He was transferred to a hospital. Lumbar puncture yielded blood-stained CSF. Urine and blood studies, including clotting studies, were normal. His past medical history was noncontributory. He had moderate meningism, was somnolent and disoriented. Spontaneous speech, comprehension and repetition were normal. He had no abnormal motor signs or alterations in reflexes. Pain sensation was intact.

Cranial computer tomography revealed fresh blood in the left lateral ventricle. A fourvessels magnification angiography displayed an arterio-venous malformation (AVM) in the atrium of the left ventricle. Feeding arteries arose from the left posterior pericallosal and posterior choroidal arteries, the draining vessel was the enlarged left internal cerebral vein.

On September 26, 1990 the AVM was removed through a left occipito-parietal craniotomy. The neurosurgeon decided on an access from the left superior parietal lobule. After having dissected the upper parietal white matter and the isthmus of the corpus callosum, he found a vascular malformation rising from the choroidal plexus of the left atrium. During the operative preparation of the malformation the partially necrotized left fornix and a portion of its (fornical) commissure ruptured.

Postoperative magnification angiography confirmed the total removal of the vascular malformation. A hemorrhage on the first postoperative day broadened out the access lesion to a cylindric necrosis measuring up to 3 cm in diameter. Following this complication the patient developed a mild right-sided hemiparesis, which had receded six months later. Elementary somatosensory perception was normal.

## General Neuropsychological Test Results

When admitted to our department (3 months after surgery), tactile-kinaesthetic object recognition (stereognosis) with the right hand was abolished. Six months later the stereoagnosis was virtually unchanged. D.C. had a right-sided homonymous hemianopsia for white light with a residual visual field of 3 degrees visual angle on the horizontal axis. When retested 6 months later, his visual field for white light was normal. The visual fields for color and form, however, remained shrunken with only 24 degrees eccentricity on the horizontal axis. Visual search was not obviously impaired; the patient searched visual targets in both hemifields up to 52 degrees eccentricity.

Initially D.C. showed a high error rate when estimating angles and matching positions of dot patterns. He had no optic ataxia. When retested 6 months later, his visual-spatial abilities were no longer impaired.

The patient obtained a percentile score of 0 in the "Zahlenverbindungstest (ZVT, Oswald and Roth, 1978), an extended German version of part A of the "Trail Making Test". When retested 6 months later, he did nearly as poor as before (percentile score 1). On the Paced Serial Addition Task (PASAT, Gronwall and Wrightson, 1974), however, he reached a percentile of 37. Thus, his information processing ability was not generally reduced. At admittance general intelligence tested with the Standard Progressive Matrices (Raven, 1956) was above average (IQ 112). He performed well on categorization tasks (Weigl Test, De Renzi et al., 1966; Modified-Card-Sorting-Test, Nelson, 1976) but did less well in a transformation task (Tower-of-Hanoi puzzle/four-disk version, Spitz et al., 1982).

We observed a striking lack of motivation in the sense that particularly his goal striving behaviour was reduced (Gollwitzer, 1992). We also suspected a lack of emotional reactivity, since the patient was particularly poor at verbalizing his emotional state.

#### Memory and Learning Ability

The patient was examined with a Memory-Test-Battery which covered a wide range of mnemonic aspects. Tests were applied at admittance to our department (3 months post surgery) and again 6 months later.

#### Working Memory (see Table I)

Initially, D.C. had a normal digit span, while free recall of a word list and the patient's performance in the Corsi block tapping test were below average. When retested, free recall of the word list remained impaired. Additional investigation of verbal working memory re-

Memory tests	3 months	9 months
Digit span	6	6
Word span (Vorländer, 1987) Short nouns Phonologically similar nouns Long nouns		5 4 3 *
Free recall of a 10-item word list	4/10 **	4/10 **
Reading span (Ehrhard, 1988)		1 **
Corsi block tapping test (Lezak, 1983)	4 *	5

TABLE I Working Memory, 3 and 9 Months Post Surgery

\*\* < Percentile 5 of normal controls.

\* Within percentile 5 to 25.

— No data available.

vealed normal spans for short and phonologically similar words whereas performance for long words was slightly reduced. Moreover, D.C. had difficulties in recalling the final words in a set of unrelated sentences, which he previously had read ("Reading span": Daneman and Carpenter, 1980; Ehrhard, 1988).

#### Verbal Memory and Learning (see Table II)

When first tested, D.C.'s performance was reduced in all tests of verbal memory and learning. Results were better in immediate free recall of a 57-unit story and in two recognition tests (California Verbal Learning Test: Delis et al., 1987; Ilmberger, 1988; Recurring Words Test, Stollmann, 1990). His performamnce was especially poor in three subtests of the highly demanding Lern und Gedächtnis-Test (LGT-3 of Bäumler, 1974; modified version: Meier, 1987). The patient must learn and remember (for about 20 minutes) some Turkish words, telephone numbers and details from a written text. When retested after six months, the general pattern of results remained the same, although D.C. now reached an average level in immediate story recall and in the Recurring words test. The recall of the 57-unit story after 48 hours remained considerably impaired.

Memory tests	3 months	9 months
Free immediate recall of a story	21/57 *	27/57
Story recall after 48 hours	4/57 **	2/57 **
Learning of 8 word paired associates	3/32 **	8/32 **
California verbal learning test (Ilmberger, 1988) Trial 5 Short delay (free recall) Short delay (cued recall) Long delay (free recall) Long delay (cued recall) Long delay (recognition)	9/16 ** 4/16 ** 7/16 ** 5/16 ** 8/16 ** 14/16 *	9/16 ** 3/16 ** 5/16 ** 4/16 ** 5/16 ** 14/16 *
Recurring words test (Stollmann, 1990)	33/56 *	42/56
LGT-3: Verbal memory (Meier, 1987)	27 **	27 **

TABLE II Verbal Memory and Learning, 3 and 9 Months Post Surgery

\*\* < Percentile 5 of normal controls.

\* Within percentile 5 to 25.

## Memory and Learning of Non-verbal Material (see Table III)

At the admittance, D.C.'s performance was deficient in a nonverbal selective reminding task (Muramoto, 1984) in which the patient should learn the position of ten small objects hidden under film cups. In contrast, recognition memory for faces was normal and for various designs (Recurring figures test: Kimura, 1963; Hartje and Rixecker, 1978) only slightly impaired. Performance in two subtests of the LGT-3 (learning and retention of a short path and of figural paired associates) was very poor. When reexamined, the patient achieved a normal level in the Recurring figures test while the results in the two subtests of the LGT-3 remained poor. D.C. was unable, despite several attempts, to recall the Rey-Osterrieth figure (Complex Figure Test; Osterrieth, 1944; Lezak, 1983) after he had copied it very accurately.

Memory tests	3 months	9 months
Complex figure test (Lezak, 1983) Copy Immediate free recall	_	33/36 0/36 **
Figural selective reminding test (Muramoto, 1984)	10/10 ** trial 7	10/10 trial 2
Recurring figures test (Hartje and Rixecker, 1978)	24/56 *	38/56
Recognition memory for faces (Warrington, 1984)	44/50	_
Recognition of 10 faces after 48 hours	10/10	
LGT-3: Visual memory (Meier, 1987)	22 *	19 **

 TABLE III

 Non-verbal Memory and Learning, 2 and 9 Months Post Surgery

\*\* < Percentile 5 of normal controls.

\* < Within percentile 5 to 25.

No data available.

# Implicit Memory

D.C. was tested with a computer-assisted mirror drawing task, where he was asked to connect as fast as possible the numbers 1 to 20, presented on the screen, with the help of a mouse turned 180 deg (Gatzweiler et al., 1990). He showed a normal learning curve saving 22 sec between trial 1 and trial 10 (mean value for normal controls 17 sec; SD = 12 sec).

In addition, we gave him the Incomplete Pictures Test (Gollin, 1960) in the modified version of Henke and Markowitsch (in preparation). Eighteen drawings of increasingly identifiable objects were presented in 10 versions (steps). The patient was asked to name the objects as soon as he recognized them. At a first presentation of the objects D.C. needed an average of 5.7 steps. When retested, the objects were recognized 2.4 steps earlier, indicating a normal priming effect (Saving of normal controls 2.5 steps; SD = 0.47).

#### Remote Memory

Autobiographical memory revealed an almost complete loss of information from the two months prior surgery. Otherwise, major personal events were preserved, although some details, particularly events of the preceding year, were no longer available. His domain-specific (professional) knowledge was by and large spared.



Fig. 1A – Sagittal MRI images in case D.C. on four contiguous sagittal cuts: 8mm/16mm left parasagittal (top left/right), midline (bottom left), and 8mm right parasagittal (bottom right). See text for details.

### Magnetic Resonance Tomography (9 months after surgery)

Serial T1-weighted (TE 20; TR SE 461) coronal, sagittal and horizontal images were performed on a Philips Gyroscan 515. The coronal sections were obtained perpendicular to the fronto-occipital (FO)-line.

The sagittal cuts illustrate the left-sided access lesion extending from the superior parietal lobule through the medial portion of the upper parietal white matter and the posterior cingulum (cingulate bundle) to the uppermost level of the thalamus (nucleus lateralis dorsalis, nucleus lateralis posterior, nuclei pulvinares?). Isthmus and part of the splenium corporis callosi are completely removed on the left side. Hence, damage to the left-sided supracommissural hippocampus (medial and lateral longitudinal striae, indusium griseum) can be derived. The right parasagittal section (Figure 1a; bottom right) makes it plausible that the right crus fornicis was spared.

The coronal sections confirm the previously described limbic and paralimbic lesions. Moreover, damage to the tail of the left caudate nucleus and to the parietal portion of the optic radiation seems likely. The left temporal horn (of the lateral ventricle) is clearly widened. The left HF, however, appears unremarkable.

# DISCUSSION

Our patient D.C. can be considered as amnesic in the sense that he exhibits an explicit memory disorder characterized particularly by an impairment of learning ability (anterograde amnesia). Other "cortical" functions such as intelligence, language, motor abilities were largely preserved.

This pattern of predominantly verbal memory and learning deficits resembles the findings we obtained for a group of 12 left-sided posterior cerebral artery infarctions with lesions centering on posterior portions of the HF (von Cramon et al., 1988). However, these patients never had a complete retrograde amnesia and all had been fully aware of the consequences of their memory disability.

One can speculate that D.C.'s poor metamemory does not reflect true unawareness of cognitive deficits but rather his lack of motivation and reduced emotional reactivity. In principle D.C. knew about his memory problems but did not pay very much attention to it. It seems plausible to relate some goal striving deficit to the large left-sided cingular lesion which involves both the posterior cingular cortex and the posterior cingulate bundle. Goal striving is behavior directed towards existing goals, and addresses questions not only of locomotion toward the chosen goal, but also of frustration, reaching a goal (Gollwitzer, 1992).

D.C.'s pattern of results in memory and learning tasks might be explained in this context, too. Recognition is mentally less demanding than free recall. Retrieving a whatever story after 48 hours is obviously more laborious than its immediate recall. Reorganizing the Rey-Osterrieth figure from memory appears more strenuous than copying it. We indeed believe that D.C. tended to avoid demanding encoding and retrieval processes. Though, the facts are not that simple as he never refused to participate in a test and always tried to end up even with difficult tasks. We should also mention that he performed normally on the PAS-AT, a rather demanding cognitive task. Nonetheless, motivational-emotional factors seem to have more contributed to his cognitive disturbances than when he had suffered a left-sided posterior cerebral artery infarction.

At the admittance D.C. was impaired not only in verbal but also in several nonverbal memory tests. When retested after 6 months, his performance had improved in most of the non-verbal tasks. For the initial period one could argue that the processing of figural material was partly impeded by his right-sided homonymous hemianopsia and visual perceptual deficits, presumably related to the left superior parietal lesion. Another explanation derives from the fact that under the splenium of the corpus callosum a number of fornical fibres decussate to the opposite side, thus constituting the commissure of the fornix (Nieuwenhuys et al., 1988). As mentioned above a portion of the fornical commissure had ruptured. Despite the predominantly left-sided lesion of the dorsal route, the interruption of commissural fornical fibres might have some detrimental effects on activities in the right-sided HF.

This case credits to the hypothesis that even unilateral damage to the dorsal route of septo-hippocampal projections interferes with normal explicit memory functions in humans.



Fig. 1B – Four coronal MRI images in case D.C. through the anterior (up left) and posterior (up right) 3rd ventricle, through the quadrigeminal cistern (bottom left), and the trigone of the lateral ventricles (bottom right). See text for details.

The question arises whether memory dysfunctions depended on the common disruption of all three pathways (cingulate bundle, supracommissural hippocampus, fornix) constituting the dorsal route or on one of its fibre components. We indeed believe that the likelihood for severe and persistent memory dysfunction increases with the number of fibre components of the dorsal septohippocampal route disrupted. This assumption does not exclude that acute lesions of single fibre components may be followed by modality-specific (or material-specific) explicit memory dysfunctions.

Impaired memory has been reported following so-called "exclusive" bilateral fornix damage. Heilman and Sypert (1977), for instance, presented an amnesic woman who suffered from a spongioblastoma in the subarachnoid space of the quadrigeminal cistern. The neoplasm arose in the hippocampal commissure or pineal body, destroying the posterior fornix bilaterally. A selective lesion of the proximal fornix is highly unlikely in this case. A space-occupying splenial tumour damaging the subsplenial portion of the proximal fornix almost inevitably injures the gyrus fasciolaris and the fasciola cinerea. These structures are the continuation of the supracommissural hippocampus. If the adjacent cingulate isthmus was also injured in this case, which is rather likely, the posterior cingulate bundles might have also been disrupted. The case Tucker et al. (1988) have published cannot be considered as a pure fornical lesion either. Besides the left-sided transection of the proximal fornix by a malignant astrocytoma the posterior HF itself was said to be injured in this case.

In this context, we should also mention the cases of Hodges and Carpenter (1991). The authors described two patients who developed anterograde amnesia following an apparently uncomplicated transcallosal-transventricular removal of a colloid cyst. Damage to the fornical columns, demonstrated on CT and MRI scans, seemed obvious. Other memory related structures were said to be entirely normal. The sagittal images of both cases (cf Figure 2 of the paper of Hodges and Carpenter, 1991) support the assumption that the septal region as well as the supracallosal hippocampus might additionally be damaged partly by the IIIrd ventricle colloid cyst partly by the transcallosal-transventricular surgical manoeuvre. Thus, the persistently impaired verbal memory in these cases may be due to a combined lesion of the septal region and at least one of the dorsal route fibre projections (i.e. the fornix). Several fornical cases have been documented in which memory was apparently intact although these reports provided insufficient neuropsychological data (Garcia-Bengochea and Friedman, 1987; Woolsey and Nelson, 1975). Woolsey and Nelson reported on a man with a metastasis of a lung tumour measuring up to 1.5 cm in diameter, located in and replacing the posterior columns of the fornix. Microscopic examination showed that virtually all of the body of the fornix was replaced by adenocarcinoma. The fimbria bilaterally contained an excess of fibrillary astrocytes, suggesting a certain amount of fibre degeneration. The absence of anterograde amnesia might have been due to the fact that a sufficient number of compensatory septo-hippocampal interconnections (cingulate bundle, supracommissural hippocampus, ventral route) remained intact. This assumption does not exclude that a more careful neuropsychologic examination might have revealed some definite memory impairment.

With respect to the anterior cingula Ballantine et al. (1967) provided abundant evidence that major destruction of these structures (including the cingulate bundles arising from the septal region) only transiently affected memory.

An ischemic destruction of the splenium and the posterior body of the corpus callosum is a rather unique event. Degos et al. (1987) published such a case. At the time of admission the (75-year-old) patient had been unable to retain questions that she had been asked five minutes earlier, nor could she describe the various homes in which she had lived over the years. Four months after the infarction her memory was apparently intact. Though, memory and learning functions had not been formally tested. Since the posterior supracommissural hippocampus is supplied by the same posterior pericallosal branches which caused the posterior callosal infarction, an additional damage to the posterior supracallosal striae is rather likely. The cingulate bundle, the fimbria-fornix system as well as the septum and the HF were said to be spared in this case.

Von Cramon et al. (1990) examined a woman with a small hemorrhage from a ruptured cryptic angioma in the left retrosplenial area. On early CT-scans blood was found in the left lateral ventricle, indicating a disruption of the left portion of the splenium. MRI scans illustrated a circumscribed lesion mainly destroying the left posterior cingulum. Additional damage to the retrosplenial cortex could not be excluded. Initially, verbal learning and memory functions were considerably impaired. Six months post-onset her test performance had normalized in all psychometric aspects. We concluded from this case that a retrosplenial disruption of the posterior cingulate bundle produces memory and learning dysfunction, but has no permanent effect.

It were Valenstein et al. (1987) who introduced the concept of "Retrosplenial Amnesia". The authors observed their patient T.R. over a period of 22 months. He developed retrograde and anterograde amnesia following hemorrhage from an arterio-venous malformation situated near the splenium of the corpus callosum. MRI studies demonstrated damage to the splenium and to a region containing the retrosplenial cortex and the posterior cingulate bundle. The fornix was anterior and inferior to the site of the maximal damage but, as the authors admitted, may have been involved. The HF, the thalamus and the basal forebrain were spared by the lesion. The authors suggested that the thalamo-cortical portion of Papez' circuit may be important in memory, and that lesions of the cingulum and retrosplenial cortex may cause amnesia by disrupting this pathway.

We would like to suggest an expanded hypothesis. It seems plausible that amnesia in patient T.R. was due to a combined disruption of the dorsal septohippocampal route including the posterior cingulum, the supracommissural hippocampus and the proximal fornix.

# **CONCLUSIONS**

There are several lines of evidence that the septo-hippocampal interconnections play a major role in the maintenance of normal functions of the HF in humans. Lesions of the septal/diagonal band complex and its interconnections with the HF seem to produce behavioral changes comparable to those following lesions of the HF itself with the exception that motivational-emotional factors seem to have a greater impact on memory and learning performance.

The likelihood for severe and persistent memory dysfunction presumably increases with the complete disruption of the dorsal (septo-hippocampal) route. Lesions of single fibre components including the fornix may not be a sufficient condition for lasting anterograde amnesia. A unilateral left-sided lesion of the dorsal route does not produce exclusively modality-specific deficits. The decussation of fibres in the fornical commissure could explain why some nonverbal memory functions were impaired in our case.

## Abstract

The interaction between the septal region and the hippocampal formation appears indispensable for the maintenance of normal memory and learning mechanisms in humans. The disruption of some combination of septo-hippocampal pathways, especially the disruption of the "dorsal route", deteriorates explicit memory functions.

The case of a 25-year-old male patient is presented who developed anterograde and to a certain extent retrograde amnesia following rupture and repair of an arteriovenous malformation in the atrium of the left ventricle. A left-sided lesion of the dorsal route involving the posterior cingulate bundle, the longitudinal striae (as part of the supracommissural hippocampus) and the fornix appeared responsible for his mnemonic deficits.

The implications of these findings for the understanding of other clinical cases, particularly those with lesions of the septal region, the anterior and posterior cingular gyrus/cingulate bundle and the fornix are discussed.

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